



## Hyperoxia may be beneficial

Submitted by Emmanuel Lemoine on Tue, 02/24/2015 - 15:41

Titre	Hyperoxia may be beneficial
Type de publication	Article de revue
Auteur	Calzia, Enrico [1], Asfar, Pierre [2], Hauser, B. [3], Matejovic, M. [4], Ballestra, C. [5], Radermacher, P. [6], Georgieff, M. [7]
Editeur	Lippincott, Williams & Wilkins
Type	Article scientifique dans une revue à comité de lecture
Année	2010
Langue	Anglais
Date	2010
Numéro	10 Suppl
Pagination	S559 - 68
Volume	38
Titre de la revue	Critical Care Medicine
ISSN	1530-0293
Mots-clés	Humans [8], Hyperoxia/metabolism/physiopathology [9], Inflammation/metabolism/physiopathology [10], Oxidative Stress/physiology [11], Oxygen Inhalation Therapy/methods [12], Oxygen/blood [13], Respiration, Artificial/methods [14], Sepsis/blood/physiopathology [15], Shock, Septic/blood/physiopathology [16], Ventilator-Induced Lung Injury/physiopathology/prevention & control [17]

Résumé en anglais	<p>The current practice of mechanical ventilation comprises the use of the least inspiratory O<sub>2</sub> fraction associated with an arterial O<sub>2</sub> tension of 55 to 80 mm Hg or an arterial hemoglobin O<sub>2</sub> saturation of 88% to 95%. Early goal-directed therapy for septic shock, however, attempts to balance O<sub>2</sub> delivery and demand by optimizing cardiac function and hemoglobin concentration, without making use of hyperoxia. Clearly, it has been well-established for more than a century that long-term exposure to pure O<sub>2</sub> results in pulmonary and, under hyperbaric conditions, central nervous O<sub>2</sub> toxicity. Nevertheless, several arguments support the use of ventilation with 100% O<sub>2</sub> as a supportive measure during the first 12 to 24 hrs of septic shock. In contrast to patients without lung disease undergoing anesthesia, ventilation with 100% O<sub>2</sub> does not worsen intrapulmonary shunt under conditions of hyperinflammation, particularly when low tidal volume-high positive end-expiratory pressure ventilation is used. In healthy volunteers and experimental animals, exposure to hyperoxia may cause pulmonary inflammation, enhanced oxidative stress, and tissue apoptosis. This, however, requires long-term exposure or injurious tidal volumes. In contrast, within the timeframe of a perioperative administration, direct O<sub>2</sub> toxicity only plays a negligible role. Pure O<sub>2</sub> ventilation induces peripheral vasoconstriction and thus may counteract shock-induced hypotension and reduce vasopressor requirements. Furthermore, in experimental animals, a redistribution of cardiac output toward the kidney and the hepato-splanchnic organs was observed. Hyperoxia not only reverses the anesthesia-related impairment of the host defense but also is an antibiotic. In fact, perioperative hyperoxia significantly reduced wound infections, and this effect was directly related to the tissue O<sub>2</sub> tension. Therefore, we advocate mechanical ventilation with 100% O<sub>2</sub> during the first 12 to 24 hrs of septic shock. However, controlled clinical trials are mandatory to test the safety and efficacy of this approach.</p>
URL de la notice	<a href="http://okina.univ-angers.fr/publications/ua8273">http://okina.univ-angers.fr/publications/ua8273</a> [18]
DOI	<a href="https://doi.org/10.1097/CCM.0b013e3181f1fe70">10.1097/CCM.0b013e3181f1fe70</a> [19]
Lien vers le document	<a href="http://dx.doi.org/10.1097/CCM.0b013e3181f1fe70">http://dx.doi.org/10.1097/CCM.0b013e3181f1fe70</a> [19]
Titre abrégé	Crit Care Med

## Liens

- [1] [http://okina.univ-angers.fr/publications?f\[author\]=4893](http://okina.univ-angers.fr/publications?f[author]=4893)
- [2] <http://okina.univ-angers.fr/pi.asfar/publications>
- [3] [http://okina.univ-angers.fr/publications?f\[author\]=14076](http://okina.univ-angers.fr/publications?f[author]=14076)
- [4] [http://okina.univ-angers.fr/publications?f\[author\]=13848](http://okina.univ-angers.fr/publications?f[author]=13848)
- [5] [http://okina.univ-angers.fr/publications?f\[author\]=14077](http://okina.univ-angers.fr/publications?f[author]=14077)
- [6] [http://okina.univ-angers.fr/publications?f\[author\]=13499](http://okina.univ-angers.fr/publications?f[author]=13499)
- [7] [http://okina.univ-angers.fr/publications?f\[author\]=13853](http://okina.univ-angers.fr/publications?f[author]=13853)
- [8] [http://okina.univ-angers.fr/publications?f\[keyword\]=991](http://okina.univ-angers.fr/publications?f[keyword]=991)
- [9] [http://okina.univ-angers.fr/publications?f\[keyword\]=13381](http://okina.univ-angers.fr/publications?f[keyword]=13381)
- [10] [http://okina.univ-angers.fr/publications?f\[keyword\]=13382](http://okina.univ-angers.fr/publications?f[keyword]=13382)
- [11] [http://okina.univ-angers.fr/publications?f\[keyword\]=13383](http://okina.univ-angers.fr/publications?f[keyword]=13383)
- [12] [http://okina.univ-angers.fr/publications?f\[keyword\]=13384](http://okina.univ-angers.fr/publications?f[keyword]=13384)
- [13] [http://okina.univ-angers.fr/publications?f\[keyword\]=12865](http://okina.univ-angers.fr/publications?f[keyword]=12865)
- [14] [http://okina.univ-angers.fr/publications?f\[keyword\]=13385](http://okina.univ-angers.fr/publications?f[keyword]=13385)
- [15] [http://okina.univ-angers.fr/publications?f\[keyword\]=13386](http://okina.univ-angers.fr/publications?f[keyword]=13386)
- [16] [http://okina.univ-angers.fr/publications?f\[keyword\]=13387](http://okina.univ-angers.fr/publications?f[keyword]=13387)
- [17] [http://okina.univ-angers.fr/publications?f\[keyword\]=13388](http://okina.univ-angers.fr/publications?f[keyword]=13388)
- [18] <http://okina.univ-angers.fr/publications/ua8273>

[19] <http://dx.doi.org/10.1097/CCM.0b013e3181f1fe70>

Publié sur *Okina* (<http://okina.univ-angers.fr>)